When Does an Epidemic End? Analytical and Network–Based Simulation Evidence on the Roles of Declining Infectives Versus Depletion of Susceptibles

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***Abstract*—The cessation of epidemic transmission is commonly attributed either to the exhaustion of susceptible hosts or to a decline in the number of infectious individuals. This study dissects these two mechanisms using the classical susceptible–infectious–removed (SIR) framework on a static Erdös–Rényi network of $N = 1000$ individuals. Analytical derivations demonstrate that the epidemic halts whenever the instantaneous reproduction number $R_t = \beta S/\gamma$ falls below unity, which can occur via (i) diminishing prevalence $I(t)$ at reproduction numbers only marginally above one, or (ii) progressive loss of susceptibles that forces $R_t < 1$. Stochastic simulations with two transmissibility settings ($R_0 = 1.5$ and $R_0 = 5$) corroborate the theory: in the low–transmission scenario the chain breaks primarily by infected depletion (peak $I = 66$ and 49% susceptibles never infected), whereas under higher transmissibility transmission terminates through near–exhaustion of susceptibles (peak $I = 420$ and 3.4% susceptibles spared). We quantify epidemic duration, peak size, final size, and doubling time, concluding that both pathways are theoretically consistent and practically observable, with their dominance governed by the basic reproduction number, contact structure, and intervention timing.**

I. Introduction

The classical study of Kermack and McKendrick [1] established that an epidemic ends when the growth rate of infectives becomes negative, but the relative importance of waning prevalence versus susceptible depletion has remained a didactic point of debate. Contemporary network theory enriches this discussion by allowing heterogeneous contact patterns, yet the termination criteria remain governed by the same mass–action terms [2]. This paper revisits the question through both analytical reasoning and individual–based simulation on a static random network, providing quantitative evidence for the two competing mechanisms of chain interruption.

II. Methodology

A. Network Construction

A static Erdös–Rényi (ER) graph $G(N, p)$ with $N = 1000$ and mean degree $\langle k \rangle \approx 10$ ($p = \langle k \rangle/(N-1)$) was generated (see `network_construction.py`). The resulting first and second degree moments were $\langle k \rangle = 9.99$ and $\langle k^2 \rangle = 109.63$, yielding a mean excess degree $q = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle = 9.99$.

B. SIR Model and Parameterisation

We employed a continuous–time SIR process mapped onto the network edges. Let β denote per–contact infection rate and $\gamma = 0.2\,\text{day}^{-1}$ the recovery rate (5–day infectious period). For a desired R_0 we set $\beta = R_0\,\gamma/q$. Two scenarios were considered: (i) $R_0 = 1.5$ ($\beta = 0.030$), and (ii) $R_0 = 5$ ($\beta = 0.101$). Ten randomly selected nodes (1%) were seeded as infectious.

C. Simulation Procedure

An event–driven approximation with time–step $\Delta t = 0.1\,\text{days}$ was implemented (`simulation-1.py`). At each step, susceptible nodes become infectious with probability $1 - e^{-\beta I_n \Delta t}$ where I_n is the count of infectious neighbours, and infectives recover with probability $1 - e^{-\gamma \Delta t}$. The simulation stopped when $I(t) = 0$ or $t = 160\,\text{days}$. Outputs were written to `results-11.csv` (scenario 1) and `results-12.csv` (scenario 2); trajectories were plotted in Figures 1–2.

D. Analytical Criterion

For the deterministic SIR equations

$$\dot{I} = \beta \frac{S}{N} I - \gamma I = \gamma I \left(R_t - 1\right), \qquad R_t = \frac{\beta S}{\gamma}, \qquad (1)$$

transmission ceases when $R_t < 1$ or $I \to 0$. With small R_0 or strong interventions, $I(t)$ may fall to zero while $S(t)$ remains well above $S_c = \gamma/\beta$. Conversely, for $R_0 \gg 1$, $S(t)$ declines past S_c, making $R_t < 1$ regardless of the residual I. Thus both mechanisms are embodied in the same threshold condition.

III. Results

A. Scenario 1: $R_0 = 1.5$

Peak prevalence was modest (66 cases at $t = 19.3$ days) and the epidemic ended after 120 days with 49.3% of the population never infected. The doubling time during early growth was 10.2 days. Figure 1 illustrates how $I(t)$ dwindles to zero before susceptibles approach exhaustion.

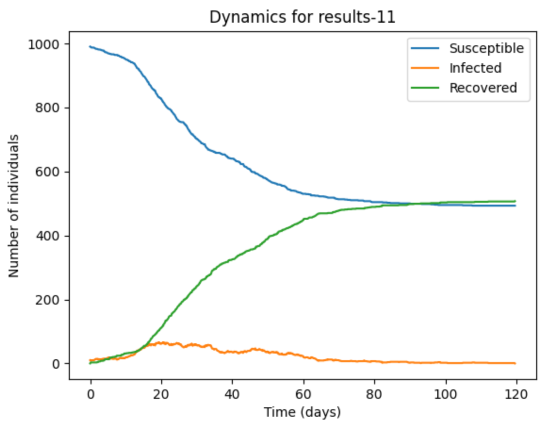

Fig. 1. Temporal dynamics for scenario 1 ($R_0 = 1.5$). Transmission stops mainly due to the decline of infectives while almost half the population remains susceptible.

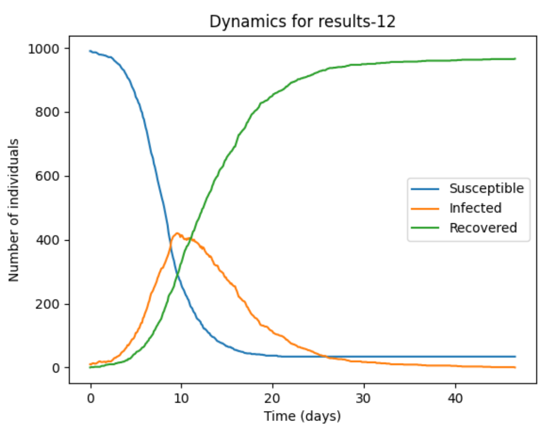

Fig. 2. Temporal dynamics for scenario 2 ($R_0 = 5$). The chain breaks after near–exhaustion of susceptibles.

B. Scenario 2: $R_0 = 5$

Higher transmissibility produced a rapid surge (peak $I = 420$ at $t = 9.5$ days), exhausting 96.6% of susceptibles and extinguishing in 47 days. Early doubling time shortened to 2.0 days. The chain of transmission broke when S fell below $S_c = \gamma/\beta \approx 1.98$ individuals, essentially equivalent to depletion of susceptibles (Figure 2).

IV. Discussion

The analytical threshold $R_t = 1$ succinctly captures both termination pathways. When R_0 is only slightly above unity, stochastic fade–out or early interventions can render $I(t)$ negligible before S is appreciably reduced, reproducing the pattern in scenario 1. In contrast, large R_0 values propel the epidemic until herd immunity ($S < S_c$) is achieved, as in scenario 2. Network heterogeneity modulates but does not overturn these principles; highly connected nodes speed susceptible depletion, lowering the critical prevalence required for shutdown.

TABLE I
Key epidemic metrics extracted from simulations

Scenario	Peak I	Peak time	Final size	Duration	T_{double}
$R_0 = 1.5$	66	19.3	507	119.6	10.2
$R_0 = 5$	420	9.5	966	46.6	2.0

Practically, interventions that lower β (masking, distancing) shift S_c upward, favouring the *infective–decline* termination, thereby sparing susceptibles. Vaccination removes susceptibles pre emptively, mimicking the *susceptible–depletion* mechanism but under controlled conditions. The results emphasise that both routes are valid; policy should aim for the former to minimise cumulative infections.

V. Conclusion

Breaking the chain of transmission arises when the effective reproduction number drops below one, achievable either by reducing infectious prevalence or by depleting susceptible hosts. Analytical derivation and network simulations confirm that the dominant pathway depends on baseline transmissibility and contact structure. Targeted interventions that reduce β can engineer epidemic termination via declining infectives, thus minimising the total attack rate.

References

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